

Society Reports.

THE NEW YORK NEUROLOGICAL SOCIETY.

*Stated Meeting, held at the New York Academy of Medicine,
on May 7, 1895.*

Dr. C. A. HERTER, Vice- President, in the chair.

Dr. J. ARTHUR BOOTH presented a case of monocular blindness without any discoverable lesion in the eye. The special point to which he directed attention was, whether after a functional blindness had lasted for a year or two, the morbid condition might not persist, finally causing congestion and inflammation? The patient, Agnes L., fourteen years of age, had been perfectly well up to about one year ago, at which time she had fallen on the ice and struck her head. Two months after this, she began to have headache, chiefly frontal. On May 5, she had come to the Manhattan Eye and Ear Hospital because of dimness of vision. Examination had revealed almost total loss of vision in the right eye, without any change in the vessels or disc. While in the hospital she had had two hystero-epileptoid attacks. Strychnine had been given freely, but without benefit. Vision in the left eye was $\frac{1.5}{20}$ ths, but could be brought up to $\frac{3.0}{20}$ ths by glasses. The field was much contracted in all directions. In a moderate light, both pupils were dilated, the right being a little larger. There was a marked loss of sensation in the right cornea, but no anæsthesia of the face, body or extremities. Colors were readily recognized by the left eye. The knee-jerks were equal, but somewhat exaggerated. The treatment by means of drugs had failed, as had also hypnotism. Various tests had been made to detect any deception on the part of the patient, but the results had been negative.

AN UNUSUAL CASE OF LEAD POISONING.

Dr. WILLIAM M. LESZYNSKY presented a man whom he had first seen about ten days ago. He had experienced difficulty in making the diagnosis at first on account of the obscurity of the etiology. The man stated that at the onset, sixteen days before, he had had fever, vomiting and malaise, but there had been no chill, and no bladder or rectal symptoms. He complained of general weakness in the lower extremities. About the same time he first noticed an aching pain in the right shoulder, and two days later, the index and the medius dropped, and the pain disappeared. A few days after this, the weakness had increased, and when first seen by the speaker, there was wrist-drop. The left arm and hand were also becoming weak, but there was no pain. Both extremities had felt numb. There was no history of exposure to cold. For five or six years he had been drinking five pints or more of beer daily. Examination showed muscular weakness—a paresis of the deltoid and upper portion of the trapezius, and the biceps group, and the extensors were paralyzed—as was also the extensor minimi digiti. There was also extreme tenderness along the course of the nerve trunk, as well as some tenderness of the muscles themselves. With such a history one would suspect lead, and there was a slight line upon the gums suspiciously like a lead line, but he was a cooper by occupation, and at first no history could be obtained of his having come in contact with lead. A provisional diagnosis had been made of alcoholic multiple neuritis, beginning in the upper extremities. The urine was then examined, and traces of lead found in it. There was no albumen or sugar. Inquiry then elicited the fact that he had occasion to close barrels containing a preparation of white lead. Electrical examination showed absolute loss of faradic irritability in the musculo-spiral on the right side, and in the extensors, and diminished faradic excitability in the other muscles that were paretic. Both knee jerks were active. Ophthalmoscopic examination was negative. This patient had also tachycardia, the pulse rate being from 116 to 120, and weak.

Dr. M. ALLEN STARR said he had recently seen a man suffering from lead palsy who had been in the habit of drinking a large quantity of water from the faucet every morning. It was believed that the water having

remained in the pipe all night had become impregnated with lead, and that this had given rise to the lead poisoning. The speaker said he had only seen one case of lead poisoning in which all the extremities had been affected nearly equally, and where there had been intense pain along the nerve trunks and muscles. This patient, a painter, had been in the hospital for fourteen months, and all this time had had the typical blue line on his gums.

The CHAIRMAN agreed in the diagnosis of lead paralysis in the case reported by Dr. Leszynsky, notwithstanding the unusual tenderness of the nerve trunks and muscles, but he was inclined to think that the fact of the man having been a steady drinker might have had something to do with precipitating these symptoms. Alcoholism certainly predisposed to attacks of lead poisoning. During the past winter, he had seen a large number of cases of severe alcoholic neuritis, and had been impressed with the fact that in a certain proportion of these the first symptoms had been pain in the joints and in the muscles about the shoulder.

AN INTRACRANIAL GUMMA.

Dr. C. E. NAMMACK presented a woman, twenty-four years of age, whom he had first seen two weeks ago. For the past five months she had had constant headache, with vertigo and vomiting in the early morning. This was followed by gradual loss of power in the right hand, followed by motor paralysis of left leg. Two months later she found that she was unable to close the left eye. There had been no injury or emotional shock, and there was no history of alcoholism or of venereal disease of any kind. She answered questions slowly, and it was difficult to keep her attention concentrated on any one subject. There was optic neuritis on both sides, with beginning choked disc in the left eye. *R.V.* was $\frac{2}{3}$ oths and *L.V.* $\frac{2}{7}$ oths, not improved by glasses. The tongue was furred on the left side only, and taste was diminished on this side. The diagnosis was an intracranial gumma, localized in the lower part of the pons on the left side. For the past two weeks she had received inunctions of blue ointment, and sixty grains of iodide of potassium, three times a day, and already the improvement was remarkable.

Dr. B. SACHS said that it was well to be cautious

about making a diagnosis of gumma in a case of this sort, solely on the strength of the improvement observed from such treatment. For example, he had seen a man of forty-five years, who had had a hemiplegic attack some years before, and in whom the administration of a large quantity of iodide of potassium had caused the entire disappearance of all the symptoms for several months. Subsequently, this man had a fatal apoplectic seizure, and the post-mortem had shown a very large glioma. In connection with every neoplasm there was a large amount of exudation, and absorption of this fluid might be responsible for the improvement following the treatment.

Dr. STARR said that if he remembered correctly, Dr. Nammack had himself recently reported to the society a similar case. Within ten days he had seen a boy of sixteen, suffering from all the symptoms of well-marked tumor of the cerebellum. When this boy was eight years of age, he had suddenly developed symptoms of tumor of the cerebellum, and had been treated for such a tumor by Dr. Seguin. Within two months after beginning the anti-syphilitic treatment, the symptoms had entirely subsided, and the boy had been apparently perfectly well up to six weeks ago, when general symptoms of tumor and eye symptoms had returned. Examination had shown the typical choked disc. Although this boy had not had syphilis, and the family history was exceptionally clear on this point, yet anti-syphilitic treatment had caused the disappearance of the symptoms of tumor.

Dr. J. F. TERRIBERRY remarked that one could get no idea of the degree of choked disc from the impairment of vision.

REPORT OF A CASE OF LEAD PARALYSIS WITH SPECIAL REFERENCE TO CYTOLOGICAL CHANGES IN THE NERVOUS SYSTEM, AND THE DISTRIBUTION OF THE LEAD.

Dr. C. A. HERTER read a paper with this title. The case forming the subject of this paper was Thomas E., twenty-six years of age, poorly nourished and undersized, who had been admitted to the City Hospital on November 23, 1894, and had died on December 12, 1894. His family history was negative. He had used alcohol moderately, but had never had syphilis. He had been a

painter for the last thirty years. In February, 1894, he had been treated in this hospital for very severe lead colic associated with double wrist-drop. With the exception of the latter, the symptoms had entirely disappeared during his stay in the hospital, and he had returned to his work. Shortly before his last admission, he began to have attacks of nausea and abdominal cramps. On admission, he complained of weakness in his arms and legs. Examination showed slight œdema of the lower extremities; apex beat diffused and rather weak; arteries distinctly atheromatous; pulse 80, weak, but regular; well marked blue line on upper and lower gums; well developed wrist-drop; complete loss of power in the extensors of the wrist on both sides, and but little power in the flexors. In the biceps of either side the power was unimpaired. There was atrophy of all the muscles below the elbow, and especially of the interossei. There was no triceps jerk. In the lower extremities, there were slight drop-foot and moderate contracture of the calf muscles. The loss of power was rather more marked on the right side. The thigh muscles reacted fairly well to faradism. The knee-jerks were feeble on both sides. No ophthalmoscopic examination was made, but the acuity of central vision was distinctly reduced. The patient was kept in bed, and received, by mistake, ten grains instead of five grains of iodide of potassium, three times a day. Two days afterward, the daily quantity of urine was 1,600 cc., it was acid, had a specific gravity of 1.016, and contained half a gramme of albumen to the litre. On December 6, there were 670 cc. of urine excreted, and it contained half a gramme of albumen to the litre; urea 14.36 grm.; uric acid .17 grm.; phosphoric acid, 4.86 grm.; sodium chloride, 1.7 grm.; pre formed sulphates, .80 grm.; combined sulphates, .20 grm. The absolutely low urea, and relatively and absolutely low uric acid were striking features. On December 7, the patient began to be drowsy during the day, and three days later he was in a muttering delirium, and the pupils were contracted and reacted but little to light. On December 10, the pupils were pin-point, the respiration was stertorous, the eyeballs prominent, the pulse feeble, and the body surface cold and clammy. Death occurred on December 12. On the last day, 410 cc. of urine were drawn by catheter. It had a specific gravity of 1.015, and contained albumen and a considerable number of granular and epithelial casts.

The ratio of uric acid and urea was 82.7; the chlorides were low, and the sulphate ratio was again exceedingly low. Blood, drawn two days before death, showed the percentage of urea to be .76, which is two to three times the normal amount. At no time were there convulsive movements.

Dr. IRA VAN GIESON described the results of the post-mortem examination. He said that attention had been paid chiefly to the cytology of the ganglion cells—in other words, to the science of these cells. It was only by such investigations that we could fully understand the changes underlying a great many nervous disorders, and particularly the toxæmias, and affections due to over-fatigue of the ganglion cells. By reference to a chart, he showed how complex was the structure of these ganglion cells, as determined by the modern methods of research. Lead poisoning, he said, should be one of the most brilliant fields for this new line of observation, as the process was so slow. At the autopsy on the case under consideration, several erosions were found in the brain. The vessels at the base were normal, as were also the cranial nerves. The viscera, with the exception of the kidneys were practically normal. The surface of the kidneys was rough and granular, the cortex was very thin, and the topography of the tubules was lost. The submaxillary glands, the thyroid and the supra-renal capsule showed no significant changes. Dr. Herter had found the epithelium of the secreting tubes in a state of marked granular degeneration, that the cells of Henle's loops were degenerated, and contained hyaline casts, and that the cells of the collecting tubes were degenerated and proliferated, and their lumen filled with degenerated cells and hyaline material. About half of the glomeruli were in a state of advanced fibrous atrophy; in the remaining glomeruli, the capsule was thickened, the capillaries compressed, and the cells of the tufts moderately increased. The tufts filled up the capsules.

The peripheral nerves had been hardened in osmic acid, and examined with the following result: In the ulnar, about one fibre in every ten was moderately degenerated; in the external peroneal, about one-fourth of the fibres were collapsed and degenerated; in the sciatic of the middle of the thigh, about one degenerated fibre was found in every ten; in the left external cutaneous, there was partial degeneration of one fibre in ten, and more complete degeneration in three fibres in ten; in

the right radial at the wrist, one fibre in twenty was degenerated; in the left plantar, one in twelve.

Portions of both enlargements of the cord were hardened in various ways, and examined in detail. By applying anilin dyes, fully one-third of the ganglion cells showed a form of degeneration which changed the internal composition of the cell, although not its shape. Comparatively few of the cells were completely destroyed. The changes observed were not apparent if specimens had been hardened in Müller's fluid, and stained with carmine. In the specimens examined, the chromophyllic granules had been broken up and disseminated throughout the ganglion cells. Such changes he thought could be considered as entirely independent of the changes observed in connection with rest and fatigue. Some of the cells showed beginning vasculatation, and others an accumulation of pigment. It was now known that the quantity of pigment indicated the age of the cell. Where the degenerative process had gone still further, the cells had been nearly destroyed. These cells had been found equally distributed in the cervical and lumbar enlargements. Fully one-third of the ganglion cells showed the mild changes first described as occurring in them.

In the sympathetic system no such changes had been as yet detected. The cerebellum in this case had been found absolutely normal. The motor zone had been particularly examined in the hand region, but as little work had been done on the normal ganglion cells here, no positive deductions could be made. It was to be remembered that in cases of toxæmia there was often a rise of temperature shortly before death, and this might produce some of the changes observed in the nervous system.

Dr. HERTER, continuing, said that the various organs or portions of them, had been oxidized as thoroughly as possible, and the lead separated from this material on platinum plates. From these plates it had been dissolved with nitric acid, and ultimately converted into a lead chromate, and weighed as such. The spleen contained .004 per cent. of lead; the kidney, .0004 per cent.; the liver, .0021 per cent.; the brain, .001 per cent.; the spinal cord, .0031 per cent. The lymph nodes of the mesentery gave no lead reactions. The soleus muscles contained .0081 per cent.; the lungs, .001 per cent.; the heart a trace, and the blood, contrary to the experience

of other writers, contained .0002 per cent. of lead. The brain, spinal cord and liver also contained small amounts of copper.

The speaker said that the case was of interest on account of the loss of power in the upper and lower extremities, and the long time the patient had been exposed to the influence of lead without exhibiting symptoms of lead poisoning, and also because of the cerebral symptoms shortly before death. The great preponderance of the lesions in the spinal cord over the peripheral nerves was, perhaps, the most striking feature of the post-mortem findings. It was to these spinal lesions that the atrophic paralyses must be referred. The changes in the lumbar enlargement were rather more marked than in the cervical enlargement.

Were the cerebral symptoms to be considered as due to the direct action of the lead on the brain? Three points should here be considered, viz.: (1) The motor areas showed no distinct change in structure; (2) the motor areas contained a small amount of lead, and (3) the fact that there were only slight changes in the cortex, which did not exclude the possibility that the cerebral symptoms were due to lead. Two cases had been reported going to show that the cerebral symptoms might be present, even without there being any lead in the brain. A number of facts gave support to the theory that these cerebral symptoms were uræmic in character. The kidneys in the case reported must have been taxed to their full functional capacity for a considerable time before death. The post-mortem findings indicated that an acute diffuse nephritis had been engrafted on a chronic nephritis. The clinical record of the condition of the urine would also seem to support this view, for just before death the elimination of urea was defective. Again, the blood drawn just before death showed a marked increase of urea. It might be assumed, therefore, that the retained urea and various leucomaines were concerned in large degree in the production of the cerebral symptoms observed shortly before death.

The evidence strongly suggested that the chronic diffuse nephritis in this case was the result of the lead poisoning. Many observations had certainly shown that this might be a cause of the small, contracted kidney. In this case, there was nothing to indicate that there was any true gouty element present. The acute nephritis was possibly induced by the irritation of the kidneys

resulting from the administrations of the large doses of iodide of potassium and subsequent liberation of lead. The spinal cord contained nearly three times as much lead as the brain. There was no significance to be attached to the small quantities of copper found in the brain.

DISCUSSION.

Dr. L. STIEGLITZ said that some years ago he had made some extensive experiments on rabbits and guinea-pigs with lead. He was gratified that the lesions described by Dr. Van Gieson confirmed his own results. Besides gross lesions in the anterior gray columns in two or three cases he had noted the vacuolation of the ganglion cells in those animals in which the paralysis had been most marked. Erb, in a paper on lead palsy, had claimed that the anatomical changes in the peripheral nerves did not settle the pathology of lead palsy—indeed, he went so far as to say that in lead palsy there might very well be first of all a functional lesion of the ganglion cells. By “functional” he meant changes so minute as not to be detected by the technique of the time; it was gratifying that the refined methods of the present day applied by Dr. Van Gieson have demonstrated these very changes.

Half of the animals he had experimented on had died of cerebral hæmorrhage. The probability that the condition of the kidney was responsible for the cerebral symptoms observed in lead poisoning, had already been urged in a paper by Westphal. It was well known that in acute poliomyelitis of children, a certain part of the paralysis was entirely recovered from, and there was no reason to doubt in these cases that the ganglionic cells of the anterior horn were affected, probably by minute lesions similar in character to those just described by Dr. Van Gieson.

The determination of lead in the different organs was a very crude method, and one should be cautious about deductions found on such determinations; an organ like the spleen, for instance, might contain a great deal of lead without functional disturbance, and the organ more sensitive to the poison, like the cord, contain very much less and still suffer to a greater degree.

He believed the pathology of lead palsy could only be decided when we had found a micro-chemical bath for lead, for we could then demonstrate the lead in the

degenerated ganglionic cells or nerve fibres, as the case might be.

He had devoted considerable time to observations on the kidneys of animals and had found both chronic and acute nephritis.

Dr. MARY PUTNAM-JACOBI thought that the facts brought out to-night showed that the lead acted, not after being deposited in an organ, but during its circulation through the organ and by interfering with the metabolism of its tissues. Thus the nutrition of the ganglion cells might be seriously depressed, and yet only a minute quantity of lead be detected in nerve tissue. The frequent association of gout with lead poisoning was another indication that the lead acted by disturbing metabolism. Nephritis was probably induced by the irritation of organic poisons thus formed, rather than by direct action of the lead on the kidneys. This view would seem to be more in consonance with our knowledge of the comparatively indifferent nature of lead.

Dr. HERTER, in closing the discussion, said, he agreed with Dr. Jacobi that the small, granular kidney was produced in cases of lead poisoning by an indirect influence on the metabolism of the body, resulting in an increased amount of work being thrown on the kidney. He had not been aware that Westphal had stated his belief that lead encephalopathy was due to the condition of the kidneys, although he knew that Oliver had accepted this view for a certain small number of cases. He did not think, however, that anyone previously had demonstrated that the blood in these cases did actually contain a marked excess of urea and allied substances.